Chapter 15

Diseases of the Urinary Tract and Kidney

John Pringle

I. RENAL DISEASE

A. Renal disease in horses

1. Acute renal failure is a sudden, theoretically reversible inability of the kidney to function in clearing nitrogenous wastes while maintaining fluid and electrolyte homeostasis.
   a. Patient profile. Acute renal failure can occur in horses of any age.
   b. Clinical findings. Signs of acute renal failure are nonspecific and are often related to concurrent disease (e.g., colitis, diarrhea, exertional rhabdomyolysis).
      (1) Complaints include anorexia, depression, weakness, and decreased athletic performance. There may be abnormal frequency or volume of urination. Edema and increased water intake can also occur.
      (2) Oliguria is a characteristic finding with hemodynamic causes, whereas polyuria may be evident with acute renal failure caused by aminoglycosides.
   c. Etiology and pathogenesis
      (1) Etiology. As in all species, the inciting cause of reduced kidney function in horses can be prerenal, renal, or post renal.
         (a) Prerenal causes are factors that decrease blood flow to the glomerulus. These factors include severe hypovolemia due to dehydration, endotoxemia, or cardiac failure; vascular injury due to endotoxin; or compromised autoregulation of renal blood flow by prostaglandin synthase inhibitors [i.e., nonsteroidal anti-inflammatory drugs (NSAIDs)]. Many compounds are considered potentially nephrotoxic, but the mechanisms are not well documented.
         (b) Renal causes directly damage the kidney tissue. Many toxins have a specific site of action, such as the glomerulus or the proximal tubules. However, there are no tests available to diagnose the site of damage, which could then lead to the early recognition and removal of toxin. Renal causes include:
            (i) Nephrotoxic medications, such as aminoglycosides, certain sulfonamides, polymyxin B, phenylbutazone or other NSAIDs, and menadione sodium bisulfite (vitamin K_3)
            (ii) Endogenous pigments, such as hemoglobin from acute intravascular hemolysis or myoglobin from a large release from muscle
            (iii) Substances in various plants (e.g., oak, wilted red maple leaves, wild onion, white snakeroot) and some heavy metals (e.g., mercury), which might be contained in some blistering agents
            (iv) Cantharidin, the toxin in blister beetles (signs of intestinal erosive disease overshadows any such accompanying toxicity)
         (c) Postrenal causes of renal failure impair the animal's ability to rid itself of the urine that has been produced. Postrenal causes in horses include mainly bladder rupture in newborn foals. Although uroliths can develop in adult horses, they less commonly cause urinary obstruction in contrast to other species.
      (2) Pathogenesis. Regardless of the cause, the common elements of acute renal failure include the accumulation of nitrogenous wastes in blood, with serum creatinine elevations above 170 mmol/L and blood urea exceeding 9 mmol/L. These changes do not occur until two-thirds to three-fourths of the nephrons are no longer functioning; therefore, lesser degrees of kidney damage do not result in detectable accumulations of nitrogenous wastes.

d. Diagnostic plan and laboratory tests
   (1) Laboratory tests
      (a) Elevated creatinine and urea reflect an inability to rid the body of nitrogenous wastes, but these results do not provide the localization of the problem or the cause. Serum electrolytes, including sodium, potassium, and chloride, are initially normal but can all decrease with diarrhea or polyuria.
      (b) Urinalysis. A urine sample should be obtained to ensure urine flow.
         (i) Urinalysis showing a urine specific gravity of less than 1.02 in the presence of clinical dehydration is suggestive of intrarenal disease.
         (ii) The color of urine, the presence of the heme pigments myoglobin or hemoglobin, and the presence of free red blood cells (RBCs) or protein can be used to indicate possible underlying causes.
         (iii) Sediment analysis normally reveals considerable mucus and calcium carbonate crystals, and casts are easily overlooked because they dissolve quickly in the normally alkaline urine of herbivores.
   (2) Renal **ultrasonography** may detect cystic or structural changes in the kidney or renal pelvis.
   (3) Nuclear medicine techniques, where available, measure the glomerular filtration rate.
   (4) Renal **biopsy** can be performed with ultrasound guidance or blindly, but, because there is the risk of serious hemorrhage, this test should be resewed for cases in which biopsy is an essential part of determining the prognosis.
e. Therapeutic plan
   (1) The correction of fluid, electrolyte, and acid–base **disorders** is essential. The amount of fluids required should be based on the state of hydration. The packed cell volume (PCV) and total proteih (TP) measurements can be used to estimate the fluid deficit.
      (a) Oral fluids (e.g., water, isotonic saline, or a balanced electrolyte solution) are usually well tolerated, except in the case of acute renal failure associated with gastrointestinal disease (e.g., colitis). Electrolytes ideally should be tailored to the requirements identified by the serum electrolyte and blood gas analysis. Generally, a balanced electrolyte solution with a bicarbonate source, such as **lactated** Ringer's solution, is sufficient. Adult horses (400–500 kg) can be given 6–8 L of warm water or electrolytes every 30–60 minutes orally until **rehydrated.**
      (b) Intravenous therapy should be resewed for patients with gastrointestinal problems.
   (2) Furosemide, **dopamine,** or both are indicated in those horses that fail to begin passing urine. These horses have the anuric form of renal failure.
   (3) Underlying diseases, such as septicemia or rhabdomyolysis, should be **treated.**
   (4) Potentially nephrotoxic drugs (e.g., NSAIDs, aminoglycosides, sulfonamides), which can be far more nephrotoxic in the presence of dehydration, **should be** discontinued.
f. Prognosis for recovery is good but depends largely on the early detection of renal failure, appropriate treatment, and the ability to adequately treat concurrent disease.
g. Prevention includes providing adequate fluid therapy when there is circulatory compromise or exposure to potential nephrotoxins.

2. Renal dysfunction in the neonate is poorly understood.
   a. Some newborn foals may have high serum creatinine levels detected shortly after birth. Although this finding may indicate a renal disorder, high serum creatinine levels can also occur because of a placental problem in the mare. In these cases, the serum creatinine should become normal within several days after birth, and the foal requires no specific treatment.
   b. Newborn foals also can have **hyposthenuric** urine (1.006) for a short period after birth, which may only indicate renal immaturity.

3. Chronic renal failure is a progressive renal disease resulting from the continued loss of nephronal function or population reduction. This disorder may be a sequela to acute renal failure. There are two broad categories of chronic renal failure in horses: glomerulonephritis and tubulointerstitial disease.
   a. **Glomerulonephritis** is immunologically mediated and is the most common form of chronic renal failure in hones.
      (1) Patient profile. This disorder can occur in horses of any breed, age, or sex.
      (2) Clinical findings. The signs noted in horses depend on the stage and severity of the renal damage. Chronic weight loss, anorexia, and polyuria with the consumption of large quantities of water usually are key findings. Also, if there is major glomerular damage, there may be dependant edema due to massive urinary protein loss, which results in hypoproteinemia.
      (3) Etiology
         (a) The glomerular lesion is caused by circulating immune complexes to viral [e.g., equine infectious anemia **(EIA)],** bacterial (streptococcal), or parasitic antigens that deposit on the epithelial side of the glomerular basement membrane.
         (b) Although less common in horses, the glomerular damage can also be the result of autoimmunity, characterized by the formation of antibodies against the glomerular basement membrane.
      (4) Pathogenesis. The pathogenesis of both types of chronic renal failure involves a decreased glomerular filtration rate in which solutes that are normally filtered and secreted by tubules are retained. There is also a loss of plasma electrolytes (e.g., sodium, chloride, phosphate), which are normally retained in the body. In glornerulonephritis, autoimmune deposits and viral, bacterial, or parasitic deposits activate the complement system, which leads to cellular influx and increased vascular permeability of the glomerular basement membrane, allowing the leakage of large protein molecules (e.g., serum albumin).
         (a) Nephrons that can still function have to increase solute filtration. This excess solute flow results in inefficient water and electrolyte handling, which leads to diuresis and an observed polyuria with a compensatory **polydipsia.**
         (b) As a result of the reduced ability of the tubules to handle water and electrolytes, there is increased sodium, chloride, and phosphate in the urine. Decreased reabsorption of bicarbonate with decreased hydrogen ion excretion may also result in acidosis.
         (c) Despite the increased filtration by the nephrons, uremia occurs, and long-term effects cause a moderate anemia, focal ulceration of oral and intestinal **mucosa,** uriniferous odor to the breath, and excessive dental tartar.
      (5) Diagnostic plan and laboratory tests
         (a) Laboratory findings
            (i) Moderate **azotemia** and isosthenuria may be evident in affected horses with normal hydration.
            (ii) Persistent proteinuria without hematuria is specific to glomerulonephritis.
            (iii) Specific urine protein testing should be performed because the routine urine dipsticks often give a false-positive result for protein in alkaline or concentrated urine.
            (iv) Hypoproteinemia or hypoalbuminemia may also be found in the serum if there have been prolonged losses.
            (v) Hypercalcemia may be present, but this finding may indicate a diet high in calcium (e.g., alfalfa).
         (b) A renal biopsy can be taken but may not be warranted because of the risk of hemorrhage and the lack of contribution to therapy and prognosis.
      (6) Therapeutic plan. There is no effective treatment for glomerulonephritis because it is usually only recognized when permanent renal insufficiency has occurred. Usually, the disease progresses, and ultimately, the horse must be euthanized.

(a) Corticosteroids may be administered to reduce the effects of the immune complex disease.
(b) Diet. Horses that are stable and not markedly affected by the clinical effects of the disease can be managed with a high-quality carbohydrate diet and reduced protein (less than 10%) in feeds.
(c) Plasma transfusions have been advocated to provide temporary relief of edema caused by hypoproteinemia.

(7) Prevention is not possible because the reasons for a specific horse developing the disease are unknown.

b. Tubulointerstitial disease

(1) Patient profile. This disease can occur in horses of any age or breed and may be related to a history of prior acute illness that caused acute tubular necrosis.

(2) Clinical findings

(a) Signs are similar to chronic renal failure of glomerulonephritis [see I A 3 a (2)], with the exception of edema of hypoproteinemia. Affected horses also have **polyuria** or **polydipsia,** but in certain management situations where water consumption is not readily observed, this may go unnoticed.
(b) On rectal palpation, the left kidney may be smaller than normal.

(3) Etiology. Tubulointerstitial disease may be a sequela to acute tubular necrosis, with reported causes in horses including vitamin K_3 administration, aminoglycoside or mercury toxicity, pyelonephritis, hydronephrosis, myoglobinuria from acute myositis, or nephrolithiasis. Often, however, the cause is not determined.

(4) Diagnostic plan and laboratory tests

(a) Laboratory findings
(i) **Azotemia** and **isosthenuria** without any clinical dehydration is evident. In tubulointerstitial nephritis, there is little protein in the urine.
(ii) Electrolyte abnormalities of hyponatremia, hypochloremia, hypercalcemia, and hypophosphatemia may be evident.
(b) Renal ultrasound can identify a renal mass or renal pelvis calculi.
(c) A renal biopsy can be performed, but this test seldom provides information regarding the cause or directs treatment

(5) Therapeutic plan. Long-term treatment is unlikely to be successful, but, because these horses are not losing protein in large quantities, they can often be managed humanely by ensuring unlimited access to water, provision of a salt block, and good-quality feed with low calcium content (no alfalfa).

(a) Any **prerenal** component to the renal failure (e.g., diarrhea, dehydration) or any acute exposure to nephrotoxic drugs or agents should be corrected.
(b) Ancillary treatment may include anabolic steroids and **B vitamins.** Periodic serum monitoring of blood gases can be done, and if plasma bicarbonate drops below 18 mEq/L as a result of acid retention, the horse can be given sodium bicarbonate (225 g/day orally).

(6) Prevention. Horses with acute renal failure, particularly of hemodynamic or toxic causes, should be treated early in the course of disease and with sufficient amounts of fluid support to prevent this permanent renal tubular damage.

4. Pyelonephritis

a. Patient profile. Pyelonephritis mainly affects female animals. However, in certain circumstances (e.g., bladder paralysis), males may also develop pyelonephritis.
b. Clinical findings. In horses, pyelonephritis is often subclinical, with the only detectable signs being frequent urination and pus in the urine.
c. Etiology and pathogenesis

(1) Etiology. Bacteria isolated from affected horses include coliforms and *Proteus* species.
(2) Pathogenesis. In horses, this disorder can follow parturition, be associated with urinary bladder atony or ectopic ureters (see II D), or may occur without any identifiable risk factor.

(a) Urine **stasis,** which occurs in ectopic ureter or bladder atony, is a recorded risk factor.
(b) The short urethra in females predisposes them to the development of ascending urinary tract infection, which leads to pyelonephritis.

d. Diagnostic plan and laboratory tests

(1) Laboratory tests

(a) Pyuria is usually a hallmark of the disease and may be accompanied by proteinuria and hernaturia. These urine changes can also be found in cystitis; however, evidence of renal involvement may be observed with systemic changes to blood samples (e.g., leukocytosis with a neutrophilia, hypergammaglobulinemia, high fibrinogen).
(b) Azotemia of renal failure may be noted but is not always present, because the infection may be restricted to the renal pelvis, may affect only one kidney, or may result in damage to less than two-thirds of the body's renal function.

(2) Renal ultrasound may be used to detect purulent debris in the renal pelvis or enlargement of the renal pelvis.
(3) Urine culture confirms the causative organism but does not indicate the extent of invasion in the urinary tract.

e. Therapeutic plan

(1) Any predisposing factor, such as ureteral ectopia or ascending urinary tract infection, should be treated. To assess the response to treatrnent, a catheterized urine sample can be submitted for culture and cytology 1 week following the cessation of therapy to ensure that the urinary tract has returned to its normally sterile condition.
(2) Catheterization. When bladder atony or paralysis is the cause, the bladder should be emptied frequently by catheterization. However, a return to normal bladder function is needed for long-term success in treatment.

f. Prognosis. The long-term survival of affected animals depends on early detection and appropriate treatment. The correction of any predisposing urinary tract abnormality that may result in continued urine stasis also influences long-term recovery.

B. Renal disease in cattle

1. Acute tubular necrosis is reported as the most common cause of renal failure in cattle in selected areas of the United States and may be related to the increased risk of plant toxicities in those regions.

a. Patient profile. Acute tubular necrosis usually affects adult cattle when related to plant toxicity, but this disease can occur in cattle of any age when associated with the administration of nephrotoxic drugs.
b. Clinical findings

(1) Complaints are nonspecific and include mild depression, anorexia, dehydration, and decreased **rumen** motility or rumen stasis.
(2) Physical examination reveals an elevated temperature, pulse, and respiratory rate.

(a) A primary disorder (e.g., sepsis, diarrhea) may be obvious, predisposing the animal to the development of acute tubular necrosis.
(b) A bleeding diathesis may be seen in uremic cattle, along with recumbency.
(c) On rectal palpation, the kidney is likely a normal size and consistency.

c. Etiology and pathogenesis. Acute tubular necrosis can be caused by decreased renal blood flow, the administration of nephrotoxic drugs, or the ingestion of nephrotoxic plants. The management systems of cattle production may expose cattle to all of these causes.

(1) Decreased renal blood flow

(a) Hypovolemia. Acute severe volume depletion may be caused by diseases such as neonatal calf diarrhea, lactic acidosis ("grain overload"), or abomasal torsion (in older cattle).

(b) **Hemodynamically** mediated diseases (e.g., endotoxemia of mastitis or metritis) can also cause decreased renal blood flow.
(c) Severe **ruminal** distention (e.g., bloat, vagus indigestion] is another cause of decreased renal blood flow.

(2) Nephrotoxic drugs can cause tubular damage.
(a) The most commonly reported nephrotoxic reaction is aminoglycoside **toxicity** from neomycin.
(b) Selected sulfonamides and the administration of outdated or excess doses of tetracyclines can also result in nephrotoxicity.
(c) Acute intravascular hemolysis in cattle (or sheep) from copper toxicity results in tubular necrosis from endogenous pigment damage.

(3) Plant toxins that result in tubular necrosis include oak (*Quercus* species), which is particularly common in the southeastern United States, and oxalate-containing plants, such as redroot pigweed (Amaranthus *retroflexus).* The effect of any nephrotoxic agent is enhanced by decreased blood volume or electrolyte (sodium, potassium) depletion.

d. Diagnostic plan and laboratory tests. Failure of renal function is usually diagnosed by laboratory evaluation because clinical signs are seldom diagnostic.
(1) Serum creatinine and urea are increased, with urine specific gravity less than 1.022.
(2) Proteinuria may be present. If the sample is analyzed rapidly before the destruction by alkaline urine, granular casts (an early finding in acute renal tubular necrosis) may be present.
(3) Dehydration is suggested by the increased hematocrit and total plasma protein.

e. Therapeutic plan
(1) Fluid therapy. The main goal of treatment is providing intravenous fluid and electrolytes to restore and maintain circulating blood volume, which ensures renal perfusion. Fluids should be isotonic, containing sodium, potassium, chloride, and calcium. Normal saline with small quantities of added potassium and calcium can be used.
(2) Other treatments include administering appropriate antimicrobial therapy (if there is ongoing sepsis), discontinuing any aminoglycoside, sulfonamide, or tetracycline therapy, and relieving any abdominal distention.

f. Prognosis. Acute tubular necrosis is a highly reversible condition if detected early and treated appropriately, particularly if the condition is related to decreased renal blood flow. The prognosis is less favorable if there is sepsis associated with the tubular necrosis.

g. Prevention of acute tubular necrosis in cattle includes avoiding the use of potentially nephrotoxic drugs and restricting access to pastures that may contain plant nephrotoxins (e.g., oak).

2. Amyloidosis

a. Patient profile. Renal arnyloidosis, although rare in horses, is sporadically diagnosed in cattle, particularly in those older than 4 years with chronic foci of inflammation. This disorder is also occasionally diagnosed in sheep and goats with paratuberculosis or visceral caseous lymphadenitis (CLA).

b. Clinical findings
(1) Complaints include ventral edema, chronic intractable diarrhea, and weight loss.
(2) On rectal palpation, the kidneys are uniformly enlarged.

c. Pathogenesis. Affected cows usually have a chronic bacterial infection (e.g., pericarditis, pulmonary abscessation, peritonitis, metritis). These bacterial infections lead to reactive systemic amyloidosis and the production of amyloid **("AA")** protein, which is a β-pleated protein that is resistant to normal proteolytic digestion. Amyloid protein is deposited on the glomeruli and results in the gross enlargement of the kidneys. The cell of origin of amyloid protein is unknown.
(1) Glomerular proteinuria results in hypoproteinemia with subcutaneous and visceral edema (e.g., ascites, pleural and pericardial effusion).

(2) Amyloid infiltration into the small intestine, resulting in gastrointestinal lymphangiectasis and edema, intestinal malabsorption, and gastrointestinal motility dysfunction, is responsible for the intractable diarrhea and weight loss.

d. Diagnostic plan and laboratory tests
(1) Laboratory tests. Blood samples reveal azotemia with persistent and massive proteinuria. The hemogram is usually normal, but fibrinogen is elevated.
(2) Liver function tests. Occasionally, there is liver involvement.
(3) Biopsy of the kidney can be used to confirm the diagnosis.

e. Therapeutic plan
(1) Dimethylsulfoxide (DMSO) administration has resulted in clinical improvement in humans, dogs, and hamsters.
(a) The exact mechanism of action is unknown, although DMSO has been shown to prevent the precipitation of Bence Jones proteins and to solubilize suspensions of amyloid fibrils.
(b) Approved uses. Because DMSO is not approved for parenteral use in food-producing animals, it should be reserved for the treatment of animals to allow for the harvesting of future genetic stock, such as semen or embryo production.
(2) Broad-spectrum antibiotics can be used to treat any underlying bacterial infection.
(3) Plasma transfusions and diuretics can be administered to temporarily alleviate signs of edema.
(4) Euthanasia. Given the grave prognosis, most animals should be euthanized when the diagnosis is established.

f. Prevention. Because amyloidosis occurs sporadically, there are no recommended preventive measures.

3. Pyelonephritis

a. Patient profile and history. Pyelonephritis usually occurs in adult dairy cows from November to May (i.e., during the time the cows are more likely to be stabled indoors). Recent urinary catheterization or artificial insemination may be found in the history.

b. Clinical findings
(1) Complaints. Affected cattle may have an acute decrease in appetite and milk production, show reluctance to walk, and may have abdominal pain that could be confused with an intestinal obstruction. Although these signs are very similar to traumatic reticuloperitonitis (TRP), affected animals resist a withers pinch (in contrast to those with TRP) and are not sensitive to pressure at the xiphoid region.
(2) Physical examination findings
(a) Urine. The urine initially has blood clots associated with short episodes of acute colic. As the disease progresses, frank pyuria may be present. Pollakiuria and hematuria are also seen.
(b) On rectal examination, the kidneys may be enlarged with a loss of normal lobulation. More chronic cases also have ureteral enlargement that can be palpated rectally.

c. Etiology and pathogenesis
(1) Etiology. In cattle, *Corynebacterium* renale can cause pyelonephritis, sometimes in outbreaks. C. renale is found in clinically normal cattle, and the organism does not survive in the environment for a long period of time.
(2) Pathogenesis
(a) Transmission occurs via mechanical means, such as tail switching, urine splashing, and the use of contaminated equipment (e.g., catheters, specula).
(b) Route of infection. When the organism gains entry, it ascends the urethra (not always bilaterally), invades the renal pelvis and medulla, and later invades the renal cortex, causing fibrosis.
(c) Manifestations of disease include:
(i) Toxemia and fever

(ii) Uremia (with extensive bilateral involvement)
(iii) Abdominal pain, caused by the obstruction of the ureter or renal calyx by pus or tissue debris

d. **Diagnostic** plan and laboratory tests are the same as for horses (see I A 4 d).
e. **Therapeutic** plan
(1) Penicillin is the treatment of choice (30,000–50,000 IU/kg every 12 hours for at least 10 days). In well-established cases, prolonged therapy for up to 6 months may be necessary. To assess the response to treatment, a catheterized urine sample can be submitted for culture and cytology 1 week following the cessation of therapy to ensure that the urinary tract has returned to its normally sterile condition.
(2) **Nephrectomy** may be necessary if the disease is unilateral.
f. Prognosis is the same as for horses (see I A 4 f).

C. Renal disease in swine

1. Patient profile. Sows recently exposed to natural breeding are at risk for developing acute pyelonephritis.
2. Clinical findings. The disease is observed in sows or gilts post breeding. Initially, some sows may have a vaginal discharge. Affected animals become ill suddenly, show profound depression and fever, and can die within 12 hours of the onset of clinical signs. Most affected sows die without premonitory signs.
3. Etiology and pathogenesis. The causative organism is commonly *Eubacterium suis*. Infection may be introduced at mating or may be residual from the previous farrowing. The relationship between mating and pyelonephritis is well established in sows.
4. Diagnostic plan and laboratory tests are the same as for horses (see I A 4 d).
5. Therapeutic plan. Sows that show signs of urinary bleeding or dysuria after breeding should be treated prophylactically with antibiotics. To assess the **response** to treatment, a catheterized urine sample can be submitted for culture and cytology 1 week following the cessation of therapy to ensure that the urinary tract has returned to its normally sterile condition.
6. Prognosis is the same as for horses (see I A 4 f).

II. LOWER URINARY TRACT DISORDERS

A. cystitis

1. Patient profile. In large animals, cystitis is sporadic and uncommon. This disorder occurs mainly in adult females and is associated with recent parturition or breeding.
2. Clinical findings
a. **Pollakiuria** and the passing of small amounts of turbid urine containing blood, pus, or both may be evident.
b. Perineal scalding with alopecia may be present if the process has been ongoing.
c. Rectal palpation may detect bladder thickening.
3. Etiology and pathogenesis
a. Primary **disease.** With the exception of cystitis caused by *Corynebacterium renale* in cattle and *Eubacterium suis* in pigs, cystitis is rarely a primary disease in large animals.
b. Secondary disease. Cystitis most often occurs secondary to urine retention in diseases such as urolithiasis, bladder atony, paralysis, late pregnancy or dystocia, and Sudan grass myelomalacia (in horses). The ascending infection is usually associated with Escherichia coli, Proteus species, or Actinomyces pyogenes.
4. Diagnostic plan and laboratory tests. The key part of the diagnosis is to collect samples aseptically for cytologic and bacteriologic evaluation. A sterile catheter should be used.

a. On sediment analysis, there are high numbers of white blood cells (WBCs), desquamated epithelial cells, RBCs, and bacteria (free or intracellular).
b. The complete blood cell count (CBC) is usually normal.
5. Therapeutic plan. The goal in treatment is to identify and correct any underlying predisposing cause of cystitis, such as bladder atony or urolithiasis.
a. Antibiotics (e.g., penicillin, ampicillin, cephalosporins, trimethoprim-sulfas) can be used as initial treatment because they are well concentrated in the urine.
b. Treatment duration should last from 10 days to 1 month. However, this may not be scientifically valid because uncomplicated cystitis in humans can be cured with a single high dose of antibiotics.

B. Urinary incontinence is an uncommon problem in large animals and is associated with neurologic diseases (e.g., sacral fractures) in all species. In horses, urinary incontinence is associated with equine protozoal myelitis (EPM), equine herpes virus type 1 (EHV-1) infection, cauda equina neuritis syndrome, and sorghum or Sudan grass intoxication (see Chapter 11). Other causes of urinary incontinence are rare and sporadic, including bladder tumors, estrogen-responsive incontinence in mares, and anatomic defects (e.g., ectopic ureters; see II D).

C. Patent **urachus** is discussed in Chapter 18 VI B. The urachus serves as a connection between the fetal bladder and the allantoic cavity, which should spontaneously close at birth.

D. Ectopic ureters

1. Patient profile. This congenital problem has been reported only sporadically in horses. However, it may simply be overlooked in other large animal species that are under less intensive observation. Although ectopic ureters have been reported in both sexes, they may be more readily detected in females because of more obvious urine dribbling.
2. Clinical findings. Affected horses show urinary incontinence from birth but may appear able to void urine normally. Urine scald is evident around the perineum, but the horse may otherwise be clinically normal. In prolonged cases, ascending urinary tract infection may ensue, resulting in pyelonephritis and signs of systemic illness.
3. Diagnostic plan
a. The clinical sign of urine dribbling from birth is usually sufficient for a diagnosis.
b. **Cystoscopic** observation of the aberrant entry of ureters to the bladder neck, urethra, or even vagina can confirm the diagnosis.
c. Retrograde urography and intravenous excretory urography have been used effectively to determine the location of the ureters' entry into the lower urinary tract.
4. Therapeutic plan. When identified, the ectopic ureters should be surgically relocated to enter the bladder. If the ectopia is unilateral and the ipsilateral kidney is hydronephrotic, it can be removed surgically.

E. Bladder rupture in horses

1. Patient profile. Bladder rupture occurs most frequently in male foals, but it has been found in mares after dystocia and in other adult horses in isolated cases. In foals, the rupture is presumed to occur before or at parturition. This disorder is also increasingly recognized in recumbent newborn foals that require intensive care and may be a complication of iatrogenic increases in abdominal pressure while lifting or moving the foal.
2. Clinical findings
a. Foals appear normal at birth, with signs of depression beginning approximately 24–48 hours after birth.
b. Mild but progressive abdominal **enlargement** develops, with fluid accumulation and a reduced interest in suckling.
c. Foals may make frequent attempts to urinate but often pass only small amount of urine. These signs of straining may be mistaken for meconium impaction, but

within several days, respiratory distress from the abdominal enlargement and severe depression from azotemia and fluid and electrolyte disturbances are evident.

d. Patent urachus may be an accompanying abnormality.

3. Etiology and pathogenesis
   a. With bladder rupture in male foals, the small diameter and increased length of the urethra allows pressure to build up within a distended bladder during foaling, causing the rupture of the dorsal body of the bladder (which is the weakest point).
   b. Some foals may also have a congenital bladder wall defect that predisposes to rupture during parturition, but there is little hemorrhage associated with the site of rupture.

4. Diagnostic plan and laboratory tests
   a. The history and clinical findings are highly suggestive of bladder rupture.
   b. Abdominocentesis to confirm the presence of urine in the abdomen can also be performed.
      (1) Methylene blue can be instilled into the bladder. Its presence in a peritoneal fluid sample confirms the presence of urine in the abdomen.
      (2) Creatine level. Demonstration of an abdominal fluid creatine level that is at least two times higher than the serum creatine level also confirms the presence of urine in the abdomen.
      (3) Calcium carbonate crystals. In adult horses, calcium carbonate crystals (normally fnund in urine) can be detected in the affected animal's abdominal fluid.
   c. Laboratory studies. Characteristic changes on a serum electrolyte panel (e.g., severe hyponatremia, hypochloremia, and **hyperkalemia**) indicate uroperitoneum. The hyperkalemia can be severe enough to cause cardiotoxicity. **Azotemia** is a predictable finding in foals.

5. Therapeutic plan
   a. Surgery. The tear or defect in the bladder requires surgical correction. However, because of the often profound fluid and electrolyte disturbances, initial correction of these metabolic abnormalities is essential before placing the animal under general anesthesia.
   b. Fluid drainage. The extravasated fluid in the abdomen, if causing severe abdominal distention, should be drained by a large-bore needle puncture to relieve the pressure on the diaphragm.
   c. Fluid therapy should be given in the form of normal saline to increase sodium and chloride levels. Dextrose (5%) should be added to help reduce the serum potassium. Additionally, foals may be acidotic and require sodium bicarbonate, which also helps reduce the serum potassium to less cardiotoxic levels.

F. Obstructive urolithiasis

1. **Obstructive** urolithiasis in ruminants is likely the most common and clinically important urinary tract disease of ruminants. Clinical disease occurs when calculi lodge in the urethra and cause urinary tract obstruction. The highest incidence of clinical signs of urolithiasis in cattle and sheep is noted during the early concentrate feeding period (i.e., fall, winter) and during cold weather when water consumption decreases.
   a. Patient profile. Clinical disease is mainly seen in castrated males and is particularly common in feedlot and range-fed steers or wethers. Although bulls, cows, heifers, ewes, and rams also form urinary calculi, these cases less often develop into a clinical problem.
      (1) The female urethra is shorter and more able to pass urethral calculi than the male urethra.
      (2) In bulls, the urethra is up to 40% larger in diameter than in a similarly aged steer; therefore, bulls are less likely to become obstructed by uroliths.
   b. Clinical findings vary with the site and completeness of urinary tract obstruction.
      (1) Partial or incomplete obstruction. Urine dribbling from the prepuce ("dribblers") with blood-tinged urine surrounding the prepuce may be evident, with white, pawdery crystals precipitating around the preputial orifice. These animals have prolonged, painfut urination and may tramp or tread when attempting to pass urine.

      (2) Complete urethral **obstruction.** Bladder rupture occurs after *48–72* hours if the obstruction is not relieved.
         (a) Inappetence, depression, and colic signs (with kicking at the abdomen) may be evident.
         (b) Treading. Steers shift their weight to opposing hind limbs (i.e., treading) and appear restless, getting up and down frequently.
         (c) Tenesmus may also be present, with palpable pulsations of the urethra and straining sufficient to prolapse the rectum.
         (d) The preputial orifice hairs are dry.
         (e) Sheep may also exhibit tail wriggling.
      (3) Other signs can include grunting and grinding of the teeth (i.e., odontoprisis, bruxism).
      (4) **Rectal** palpation may reveal a large and tightly distended urinary bladder.
   c. Etiology and pathogenesis. The precipitation of urinary solutes around a nidus leads to the formation of calculi. This metabolic disorder is a combination of dietary, endocrine, and climatic factors.
      (1) Nidus formation. Factors involved in nidus formation include the administration of estrogen implants or the consumption of estrogenic feeds, vitamin A deficiency, or other factors that result in excessive urinary tract epithelial desquamation.
      (2) Urinary solute precipitation occurs for several reasons, including:
         (a) increased phosphate or carbonate calculi formation in the presence of the alkaline urine of herbivores
         (b) Increased concentration of urine solutes as a result of water deprivation in cold weather
         (c) Heavy fluid loss, which may occur in hot weather
         (d) Excessive mineral intake (which often occurs in feedlots), particularly with respect to a high phosphate intake
      (3) Mucoproteins in the urine act as cementing agents to solidify the solutes that have precipitated around the nidus. Therefore, increased mucoprotein favors calculi formation. Heavy-concentrate and low-roughage feeding and the pelleting of rations (common practice in most feedlot feeding regimens) greatly increases the quantity of mucoproteins in the urine.
      (4) Calculi. Cattle usually have single, hard, discrete calculi, but there can be up to *200* calculi present in an individual animal's urinary tract
         (a) **Obstruction** location
            (i) Cattle. Stones most often cause obstruction at the distal portion of the sigmoid flexure of the penis. There is a natural stricture at this site, which is where the retractor penis muscles attach.
            (ii) Sheep and goats tend to have fine, sand-like calculi, which are located throughout the urinary tract but most often block the vermiform appendage.
            (iii) With massive urolithiasis, obstruction may occur anywhere along the urethra in both cattle and sheep.
         (b) Types of calculi. Although several crystal types have been found in ruminant uroliths, the two main types are magnesium ammonium phosphate and silicate uroliths.
            (i) Magnesium ammonium phosphate calculi are found most commonly in feedlot cattle and sheep fed high-concentrate and low-roughage rations. These calculi are highly insoluble in alkaline urine (pH of 8.5–9.5); thus, they precipitate readily in the normally alkaline urine of herbivores. These calculi are usually small, smooth, and soft, with a high recurrence because there are many present.
            (ii) Silicate calculi occur in range-fed animals in the Great Plains regions, with grazing on mature prairie grasses or wheat or oat stubble (which can contain up to *2%* silica). Water in these areas can also be high in silicates. Silicate calculi are rough and hard, usually forming only a single calculus. Given the high level of silica in both diet and water,

there can be outbreaks of urinary tract obstruction resulting from this calculi at any time of the year in any age and gender animals.

(c) Sequelae of hrolithiasis include the rupture of the urethra, rupture of the urinary bladder, or both.

(i) Urethral rupture. The calculus lodges in the penile urethra, usually at the sigmoid flexure, and causes pressure necrosis of the urethral wall. Urine leaks into the subcutaneous tissue around the penis and accumulates in the subcutaneous connective tissue along the prepuce, resulting in extensive edema along the abdominal floor (extending from the sigmoid flexure to the umbilicus). Usually, the leakage of fluid relieves the acute pain of urinary bladder distention, but over time, this fluid can cause toxemia and tissue necrosis with sloughing of the skin of the ventral abdomen.

(ii) Bladder rupture. Abdominal pain is no longer present, and there is bilateral fluid-filled distention of the abdomen (a "pear-shaped" abdomen). In contrast to urethral rupture, there is little or no detectable ventral edema in the preputial or umbilical region. On rectal examination, the bladder is not palpable.

d. Diagnostic plan and laboratory tests

(1) The clinical examination often is sufficient to make a diagnosis of either urethral or bladder rupture.

(a) Urethral rupture

(i) The ventral abdominal edematous swelling that is associated with the prepuce caudally to the level of the scrotum, accompanied by pain at the sigmoid flexure, is usually sufficient to make the working diagnosis.

(ii) In sheep and goats, the vermiform appendage is usually blocked with sabulous material. Examination of the penis tip often reveals a turgid cyanotic vermiform appendage. Blockage further proximal in the penile urethra is usually present.

(b) Bladder rupture

(i) In the patient with abdominal swelling, the five "Fs" of abdominal distention should be considered: fat, fluid, feces, fetus, and flatus. A fluid wave can usually be balloted across the abdomen, and centesis of the abdomen with a large-bore needle readily yields a large amount of clear, acellular fluid.

(ii) Palpation of the penis at the sigmoid flexure may identify the site of obstruction, with pain induced on manipulation of the region.

(iii) On rectal palpation, the urinary bladder is usually nonpalpable. Although the abdomen is filled with fluid, this cannot be determined by per rectum palpation.

(2) Laboratory tests

(a) Serum biochemistry reveals an azotemic animal with a marked reduction in serum sodium and chloride. Potassium, however, does not increase markedly in ruminants with bladder rupture.

(b) An abdominocentesis fluid sample can be used to confirm uroperitoneum (see II E 4 b).

e. Therapeutic plan. The goals of treatment are to reestablish patent urethra and correct fluid, acid–base, and electrolyte imbalances.

(1) Cattle

(a) Medical therapy

(i) For early cases of urethral obstruction in which urethral or bladder rupture have not occurred, it is possible to attempt medical therapy by using tranquilizers (acepromazine at 20–40 mg/500 kg intramuscularly), smooth muscle relaxants, or antispasmodics (e.g., dipyrone). These agents can induce relaxation of the retractor penis muscle, which allows the sigmoid flexure to straighten, producing a wider, straighter urethra. Some reports suggest a 70% effectiveness in early cases.

(ii) If there is no urine passage within 6 hours, these medications can be repeated, but surgery may be required. Rectal examination to assess bladder size and turgor can be used to assess the need for surgery.

(b) Surgery. In the case of urethral or bladder rupture, surgical intervention (under epidural anesthesia) is required.

(i) A low urethrotorny at the distal part of the sigmoid flexure can be performed to expose and remove the calculus, suturing the incision site if the stone has not caused extensive necrosis.

(ii) A high perineal urethrostomy should be performed if local cellulitis or necrosis is present. The penis is transected proximal to the site of blockage and anchored to the skin. The more proximal urethra can be probed for evidence of additional calculi, but a urethral diverticulum at the level of the ischial arch usually prevents catheterization into the bladder. Tears in the bladder wall in bladder rupture usually heal spontaneously without requiring abdominal surgery.

(iii) In both urethral and bladder rupture, systemic antibiotics post surgery are advised. The correction of fluid and electrolyte losses with isotonic sodium chloride is indicated but is seldom performed in field situations. Animals with urethral or bladder rupture should be sent to slaughter as soon as they are no longer uremic.

(2) Sheep and goats

(a) Massaging the vermiform appendage free of the sandy debris should be attempted, but usually, the vermiform appendage needs to be amputated.

(b) Catheterization. Sabulous debris in the more proximal penile urethra can be flushed out by passing a catheter up the penile urethra and instilling small amounts of saline periodically.

(c) Surgery

(i) Urethrostomy (as performed in steers) may be indicated if other treatments fail. Even after establishing urethral patency, the bladder may not spontaneously empty immediately because of chronic distention and atony.

(ii) In the cases of urethral rupture with urine leakage in the subcutaneous tissues, small linear incisions in the overlying skin can be made to drain the urine that has collected and reduce the risk of extensive skin slough.

f. Prognosis. The survival rate for urethral rupture is approximately 90%, but for bladder rupture, the survival rate is 50%.

g. Prevention. Many dietary and management factors can affect the formation of urinary calculi and subsequent obstruction.

(1) Diet

(a) For animals with phosphate or magnesium ammonium phosphate calculi, the diet can be assessed to ensure a calcium to phosphorus ratio of 2:1. Adding ground limestone to the diet can help avoid precipitation of excess phosphate in urine. Urine pH can be acidified (using ammonium chloride in the feed), increasing the solubility of the calculi.

(b) For range-fed animals with silicate calculi, a common method of reducing problems with urinary blockage is to pasture only females or bulls on the high-risk pastures. Calculi still form but seldom result in urinary obstructive problems.

(2) Adequate water should be provided, particularly in cold weather when water sources may freeze.

(3) Increasing salt intake in the diet by up to 4% can also reduce calculi-related problems. Increased dietary salt forces diuresis (which prevents the concentration of urinary solutes). Furthermore, in the case of phosphate or magnesium ammonium phosphate crystals, sodium causes chloride to displace the magnesium and phosphate, preventing these minerals from being deposited around nidus of the calculus.

(4) Delaying castration of steers until after 6 months of age can allow the development of a larger urethral diameter, but this delay may not be practical in range or feedlot animals.

(5) Adequate vitamin A intake reduces nidus formation, and estrogenic implants can be avoided to reduce the mucoprotein content in the urine.

2. Obstructive **urolithiasis** in horses
   a. Patient profile. Cystic calculi (stones in the bladder) are not common in horses and seldom cause acute clinical signs of obstruction. Some males may develop stones that lodge in the urethra.
   b. Clinical findings. Persistent hematuria (or post-exercise hematuria) is often the only clinical sign. Otherwise, horses with cystic calculi can have mild recurrent colic, urine scalding of the perineum, stranguria, dribbling urine, or pollakiuria. Weight loss and a stilted gait have also been reported. These bladder stones are usually readily palpable on rectal examination.
   c. Pathogenesis. Less is known about the formation of these calculi in horses than in ruminants, but the factors are likely similar because horses also have alkaline urine, which favors the deposition of carbonate and phosphate crystals. Calculi are usually solitary, large, and composed of calcium carbonate or phosphates. They tend to develop near the neck of the bladder.
   d. Diagnostic plan and laboratory tests
      (1) In addition to clinical findings, **cystoscopy** or ultrasound can be used to demonstrate or suggest the presence of a stone. Occasionally, calculi can be felt with a urinary catheter.
      (2) Urinalysis reveals crystals, as well as free RBCs and WBCs. Concurrent cystitis is also a common finding.
   e. Therapeutic plan. The stones can be removed surgically by either an abdominal approach or via urethrotomy in male horses. In mares, some stones can be removed manually through the urethra. Electrohydraulic lithotripsy has been used successfully in shattering the stones in situ for ease of removal.
   f. Prognosis. Horses that have had cystic calculi may have problems with chronic cystitis even after stone removal, and the calculi may recur.
   g. Prevention. In selected cases, diet supplementation with urinary acidifiers has helped prevent calculi formation.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. Which one of the following statements regarding pyelonephritis is true?

(1) Cows that are housed during the winter develop pyelonephritis as a result of the infection by *Ureaplasma laidlawi* during natural breeding or artificial insemination.
(2) In hones, pyelonephritis is a sporadic occurrence with infection by coliforms such as *Proteus* species and is associated mainly with breeding trauma or urine pooling in mares.
(3) In pigs, the organism Eubacterium suis is a key cause of pyelonephritis, and clinical signs are insidious with pyuria and chronic weight loss.
(4) Sows develop pyelonephritis as a result of *Eubacterium suis* infection from recent farrowing or natural mating, with initial clinical signs highly different from those noted in cows or horses.

2. Which one of the following statements regarding horses with chronic renal failure is correct?

(1) Hypercalcemia may be present, but it appears to be dependent on diet because calcium levels can return to normal levels in low-calcium diets.
(2) In glomerulonephritis, the glomerular lesion is caused by autoimmunity to the glomerular basement membrane, or less commonly, circulating immune complexes to viral or bacterial (e.g., streptococcal) antigens.
(3) Chronic renal failure in horses is most commonly tubulointerstitial, rather than glomerulonephritis.
(4) When recognized, glomerulonephritis in horses is best treated with corticosteroids to reverse the immunologic damage to the glomerular basement membrane.

3. Acute renal failure in ruminants can be associated with which of the following?

(1) Neonatal calf diarrhea, abomasal displacement, and lactic acidosis ("grain overload") can be causes of acute renal tubular necrosis in cattle with their associated profound volume depletion.
(2) Although arninoglycosidescan induce nephrotoxicity in all species, ruminants are usually spared from this risk because they rarely receive such drugs.
(3) Administration of outdated or excess doses of tetracyclines has resulted in renal failure in cattle.
(4) Copper toxicity in cattle and sheep causes acute renal tubular necrosis due to the cupric ion damage to tubular epithelium.
(5) Plant toxins that result in renal tubular necrosis in ruminants include oak (*Quercus* species) and Russian knapweed (*Centaurea repens*).

4. Which one of the following statements regarding urinary tract disorders in large animals is correct?

(1) Renal amyloidosis occurs with approximately equal frequency in both horses and cattle, with signs of ventral edema, chronic intractable diarrhea, and weight loss, with the kidneys often uniformly enlarged on rectal palpation.
(2) Cows affected by renal amyloidosis seldom have any other concurrent disease.
(3) Ectopic ureters in large animals is a congenital problem with signs of urine dribbling from birth. Although this disorder has been reported mainly in horses and in both sexes, it may be more readily detected in females with more obvious urine dribbling.
(4) Ectopic ureter of large animals is usually only corrected for aesthetic reasons because, apart from urine scald, the animals seldom have any other associated complications.

5. Which one of the following statements regarding foals with bladder rupture is correct?

(1) Bladder rupture occurs most frequently in female foals after dystocia or is otherwise most common in either sex foal that is stepped on by the dam.
(2) Foals appear normal at birth and within 6–12 hours become severely depressed because of azotemia and peritonitis.
(3) Foals develop progressive abdominal distention and show signs of straining, which may be mistaken for meconium impaction.
(4) If untreated, a main cause for mortality is the progressive azotemia.
(5) Tests to help confirm the presence of urine in the abdomen include the instillation of a positive contrast (such as barium) into the bladder and the demonstration of its presence in the abdomen either by radiography or a peritoneal fluid sample.

6. Which one of the following statements regarding the prevention of urolithiasis in ruminants is coned?

(1) For phosphate or magnesium ammonium phosphate calculi, the diet should have a calcium to phosphate ratio of 2:1, possibly by adding ground limestone to the diet, which can help avoid the precipitation of excess phosphate in the urine.
(2) For silicate calculi, the urine pH can be acidified using ammonium chloride in the feed because these calculi are also more soluble in acid urine.
(3) For silicate calculi that occur in range-fed animals, pasturing only females on the high-risk pastures is advised because the hormonal differences in the cows result in less risk of calculi formation.
(4) For urolithiasis of most types of calculi, the dietary salt intake should be reduced to prevent excess solutes in the urine.
(5) Delaying the castration of male animals until after 6 months of age can reduce the problem of urinary obstruction by stones because the testosterone influence prevents nidus formation.

7. Which one of the following statements regarding cystic calculi of horses is true?

(1) Clinical signs of bladder stones in horses are similar to those in ruminants, with blockage of the urethra the main clinical sign.
(2) The main type of stone in horses is silicate calculi, which results from high silicic acid on certain pastures and oat feedstuffs.
(3) Cystic or bladder stones are usually best diagnosed by urine crystal analysis.
(4) Finding high numbers of calcium carbonate crystals in the urine of horses without signs of urinary tract disease does not necessarily suggest the presence of a bladder stone.

ANSWERS AND EXPLANATIONS

1. The answer is 4 [I C 2]. Sows may die without premonitory signs. The causative organisms are coliforms and *Corynebacterium* renale. The high risk factors associated with pyelonephritis are commonly bladder paralysis, urine stasis, or ureteral ectopia. Signs in pigs are usually peracute with death.

2. The answer is 1 [I A 3 a (5)]. Hypercalcemia may be present, but it appears to be dependent on diet because calcium levels can return to normal levels in low-calcium diets. In glomerulonephritis, the glomerular lesion is caused by autoimmunity to the glomerular basement membrane or, more commonly, by circulating immune complexes to viral or bacterial (e.g., streptococcal) antigens. Chronic renal failure in hones is most commonly glomerulonephritis, rather than tubulointerstitial. Although corticosteroids have been advocated, they do not reverse the damages to glomeruli.

3. The answer is 3 [I B 1 c]. The administration of outdated or excess doses of tetracyclines has resulted in renal failure in cattle. Diarrhea and grain overload can induce volume depletion, but it usually requires a torsion of the abomasum for similarly severe vascular compromise. One of the most commonly drug-associated nephrotoxicities in the United States is from neomycin. Hopefully, this will soon be only a historical note with increased vigilance and care in medicating food-producing animals. Acute intravascular hemolysis that results in tubular necrosis from endogenous pigment damage. Although oak is toxic, ruminants can safely graze on pastures with Russian knapweed (see Chapter 11).

4. The answer is 3 [II D 1, 2]. Although ectopic ureter has been reported mainly in horses and in both sexes, it may be more readily detected in females with more obvious urine dribbling. The clinical signs are appropriate, but horses rarely develop renal amyloidosis. Cows affected by renal amyloidosis often have a chronic bacterial infection (e.g., pericarditis, pulmonary abscessation, peritonitis, metritis), leading to reactive systemic amyloidosis. Associated complications usually include urine reflux, the formation of hydroureter, and the predisposition of the animal to ascending urinary tract infection.

5. The answer is 3 [II E 2]. Foals develop progressive abdominal distention and show signs of straining, which may be mistaken for meconium impaction. Bladder rupture is more common in males, and the damage from the dam is not a recorded high risk factor. Signs usually take 24–48 hours to develop after birth, and peritonitis is not a big concern because the urine is sterile. Untreated animals get life-threatening hyperkalemia and severe electrolyte imbalances. Barium would be contraindicated and would cause an intense peritoneal inflammation.

6. The answer is 1 [II F 1 g]. For phosphate or magnesium ammonium phosphate calculi, the diet should have a calcium to phosphate ratio of 2:1. Silicate stones are not solubilized by an acidified pH change. Calculi still form in females but seldom result in urinary obstructive problems in cows. Salt in the diet, up to 4%, can reduce calculi-related problems by a forced diuresis that prevents concentration of urinary solutes. The development of a larger urethral diameter, not the influence of testosterone, reduces the risk of urinary obstruction.

7. The answer is 4 [II F 2]. Finding high numbers of calcium carbonate crystals does not necessarily suggest the presence of a bladder stone. Signs in horses are persistent hematuria or post-exercise hematuria. Horses have alkaline urine that favors the deposition of carbonate and phosphate crystals. Bladder stones are readily palpable on rectal examination, or they can be detected by cystoscopy or ultrasound.